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CASE REPORT

Ictal catatonia as a manifestation of *de novo* absence status epilepticus following benzodiazepine withdrawal

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To describe ictal catatonia as a manifestation of *de novo* absence status epilepticus following benzodiazepine withdrawal. Ictal catatonia was documented by concurrent EEG recordings. A catatonic syndrome, first diagnosed as a psychogenic reaction, was found to be an ictal event by EEG recording. De novo absence status and benzodiazepine withdrawal should be considered when a catatonic syndrome suddenly occurs in elderly patients.

Key words: ictal catatonia; benzodiazepine withdrawal; *de novo* absence status.

INTRODUCTION

Ictal catatonia, as a manifestation of non-convulsive status epilepticus, has been described only anecdotally[1–4]. There have been only a few adequately documented ictal catatonia cases seen with concurrent epileptic discharges in the EEG[5]. Further, most of the reported cases have had a previous history of psychiatric illness or epileptic seizures. We report a case in which ictal catatonia occurred *de novo* as a result of benzodiazepine withdrawal in an elderly patient without previous history of either psychiatric or epileptic illness. This case presented a serious challenge to a correct diagnosis, because catatonic syndrome in the setting of an unremarkable prior medical history and advanced age misled us to consider a psychogenic or non-organic origin for the patient's symptoms.

CASE REPORT

A 78-year-old man was referred for psychiatric assessment because of mental confusion. He was hospitalized for prostatic hypertrophy two months before the referral. At the time of examination, he presented a one week history of episodic mutism, alternating with outbursts of psychomotor agitation, manifested by violent attacks against the nursing staff. He remembered well what he had said and what was said to him during such excited states. A scrotal infection accompanied by high fever, promptly controlled by antibiotics, preceded this episode of mental aberration. A neurologic consultant diagnosed it as a psychogenic reaction and recommended an interview with a psychiatrist. On examination, the patient appeared awake, but had a fixed and glassy-eyed stare. Occasionally, upon repeated questioning, the patient would utter scanty, fragmented, but appropriate words, in a whisper. There were no focal neurological signs. However, he exhibited a waxy flexibility, maintaining his extremities in an indefinite and passively placed bizarre posture. He had no previous history of either epilepsy or psychiatric illness. The EEG on the day of the examination showed continuous bilateral spike and wave discharges (Fig. 1A). A detailed interview with his wife revealed acute discontinuation of long-standing benzodiazepine treatment upon admission. He had been receiving nitrazepam 10 mg/day and diazepam 10 mg/day for several decades. His complete blood count, serum electrolytes, blood sugar and calcium, arterial ammonia, liver function tests, and computed tomography scan were normal. Administration of diazepam 10 mg/day was renewed. Upon this partial

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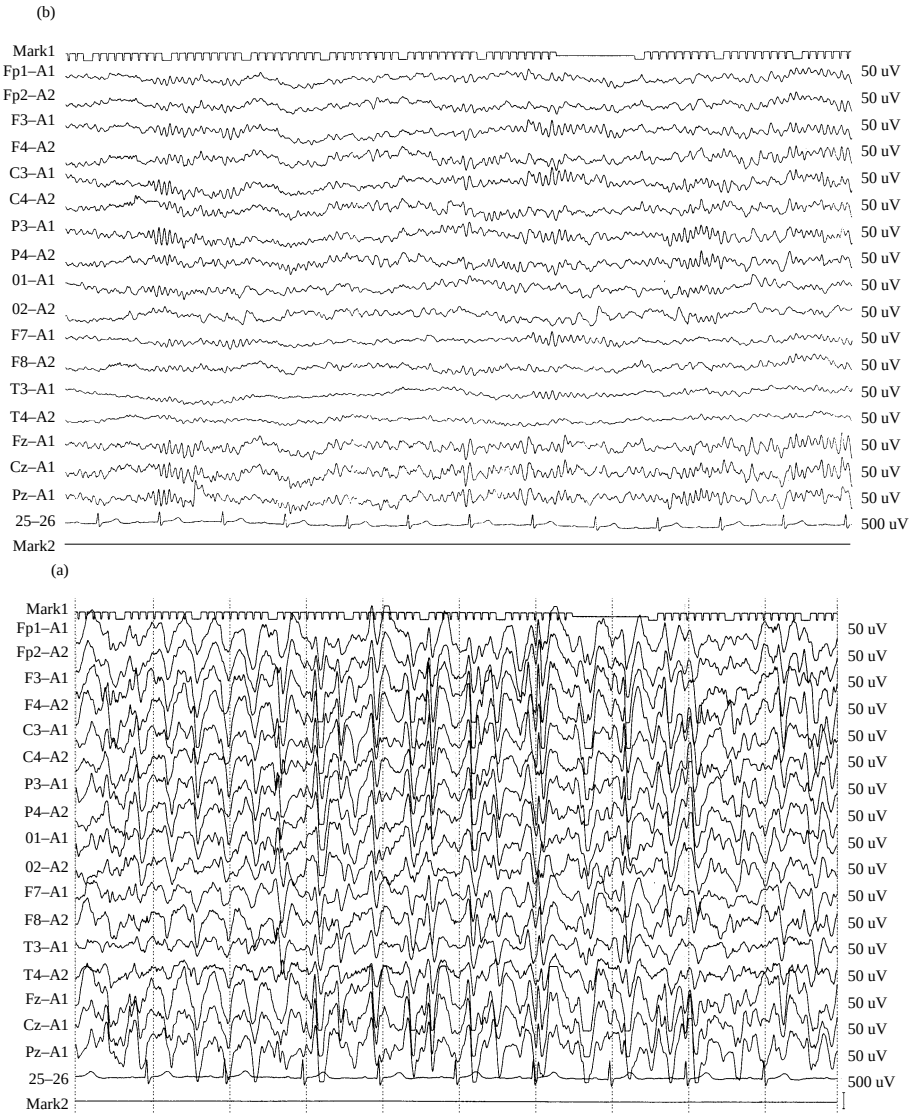

Fig. 1: (a) EEG during ictal catatonia showing bilateral pseudorhythmic 1.5–2-Hz spikes and waves. (b) Repeated EEG, after the patient had improved, showed no abnormalities.

re-administration of a benzodiazepine, the abnormal behaviour, including the catatonic symptoms, disappeared completely. The patient became alert, oriented, and acted appropriately. A second EEG (Fig. 1B) was normal. The absence status did not recur.

DISCUSSION

Although the association between epilepsy and catatonia has been pointed out repeatedly in a few case reports[3, 5] as well as in several review articles, including the original work of Karl Ludwig Kahlbaum[6, 7], an uncontested documentation of ictal catatonia with corresponding EEG findings has been rare. However, this rarity could be more apparent than real, because the peculiarity of partially preserved awareness in a catatonic syndrome, which is quite different from common delirium in the elderly, would obscure the possible organic origin from a clinician unaccustomed to such a state, as in the current case. Without EEG recordings, the ictal catatonia would remain unrecognized and could be easily dismissed as a psychological reaction.

Recently, a particular subtype of absence status epilepticus has been recognized, which appears *de novo* in middle or advanced age[8, 9]. This subtype of '*de novo*' absence status of very late onset exhibits several distinct clinical features. Firstly, it is often triggered by various exogenous epileptogenic factors, including benzodiazepine withdrawal. Thomas *et al.* [9] stressed that *de novo* absence status epilepticus was

an uncommon but important complication of benzodiazepine withdrawal syndrome, particularly in elderly persons. A careful interview with our patient's wife revealed a longstanding uptake and an abrupt discontinuation of benzodiazepines. The second salient feature of this subtype of absence status is psychopathological. While complex partial status epilepticus cases often manifest themselves as protracted confusional or acute psychotic episodes, the representative type of absence status epilepticus, typically starting in the second decade of life, usually takes on the characteristic of a simple dullness of consciousness, resulting in a simple worsening of behaviour without productive symptoms[10]. In contrast, *de novo* absence status in later life usually exhibits abundant productive symptoms, including catatonic syndrome.

There have been only scattered reports of ictal catatonia so far. Except for a brief reference in several articles, detailed documentation with concurrent ictal EEG recordings has been exceptional. Episodes of ictal catatonia, especially those which occur *de novo* in elderly persons without a related medical history, are prone to misdiagnosis. The first diagnosis of the second case of Lim *et al.* [5] was a severe psychotic depression. As a result, the psychiatric consultant recommended electroconvulsive therapy. The case of Gomez *et al.* [3], brought to the emergency room in a catatonic stupor, was assessed as having an acute exacerbation of psychosis and was given high potency neuroleptics which only worsened the patient's condition. This episode turned out later to be absence status. In the current case as well, the neurologic consultant diagnosed it as a psychogenic reaction and recommended a psychiatric interview. We emphasize that *de novo* absence status should be considered in elderly patients with a sudden onset of catatonia, especially when a benzodiazepine withdrawal syndrome cannot be ruled out.

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